Without any reference to the therapeutic indications of santonin, the writers cite the following case to show the dangers of its improper use, even when much less than the customary dose is used.

REPORT OF CASES

CASE 1. — A somewhat undernourished girl, seven years of age and weighing forty-five pounds, who has usually been in fair health except for a moderately severe attack of influenza five years before, and mild chronic tonsillitis at the time, was discovered to have pin-worms (*Oxyuris vermicularis*) and was given a preparation of santonin and podophyllin, called "worm candy" and sold to the general public by a well-known chain drug-store company. The mother thought this a safe medicine to use because, previously, she had given it to another child, on a physician's advice, and there had been no untoward results. On May 6, at bedtime, two "candies" (later ascertained to contain one-fourth grain santonin each) were taken by this girl on an empty stomach, and the next morning two more. (Total amount of santonin ingested was one grain, the printed directions having allowed 2¼ grains for one of her age.) No food was taken all this day, and a laxative at night produced a fair bowel movement. On the afternoon of May 7 the urine became dark-colored, and the child complained of seeing yellow. That night she became severely ill, had colicky pains in the abdomen, nausea and trembling, and the next day she vomited several times and was too weak to sit up. On examination, the following findings were noted: The patient was in a peculiar semistupor and complained somewhat of pains in the abdomen. There was a marked generalized jaundice, sweating, and a coldness of the skin. The pulse was slow, regular and soft, and the breathing slow. The blood pressure was 90/55. The throat was negative, except for chronic tonsillitis; the pupils reacted normally, heart tones were slow, regular and distant, and the lungs negative. The abdomen was tender to touch, particularly on the right side, the right rectus muscle being much more spastic than the left; the liver was enlarged to one inch below the costal margin in front, and the spleen and kidneys were not palpable. Urinalysis revealed: dark orange color, moderate turbidity, slight trace of albumin, no sugar, numerous blood cells, no casts, no pus cells, large amount of amorphous material, some uric acid crystals, reaction acid, Gmelin's test for bile negative, Huppert's test for bilirubin negative, while Schlesinger's test for urobilin showed a very slight fluorescence, and Hay's test for bile acids negative. The Van den Bergh test, made by Dr. Paul Christman of Sacramento, showed a biphasic reaction, and the indirect quantitative test revealed 0.8 milligram bilirubin per 100 cubic centimeters of blood. On supportive and eliminative treatment the patient recovered, most of the symptoms leaving in about four days, and the jaundice in seven; but tenderness to pressure over the liver and slight spasticity of the right rectus remained for about two weeks.

* * *

CASE 2.—A slightly underweight boy, nine years of age, who had been fairly healthy except for tonsillitis occasionally and chicken-pox and whooping-cough some years before, was given the same preparation of santonin as in the above case. The mother thought that she had seen pin-worms around the child's anus and administered the treatment without consulting a physician. The child took two "candies" on the morning of May 8, had no food all day, took two more that night, and then two more the next morning, with no food till the afternoon of the second day. (Total amount of santonin ingested was 1½ grains; printed directions allowed 2½ grains for one of his age.) No cathartic nor enema was administered, but the bowels moved naturally on the night of May 8. That same night the child became ill, and next morning the mother noticed that his sclerae were yellow and that the urine was dark orange. Soon the entire skin became jaundiced and there was giddiness, trembling, cold sweat, vomiting, followed by stupor. Physical examination revealed a very sick child—toxic, in a stupor, and markedly jaundiced. The skin was cold and damp, there was a side-to-side rolling of the eyeballs, and a twitching of the hands and arms. The pulse was slow and soft, and the heart-tones feeble. Later the liver and spleen were both determined to be enlarged, but the kidneys were not palpable. Urinalysis: Dark brown color, moderate turbidity, no sugar, large number of blood cells, no casts, no pus cells, large number of crystals, large amount of amorphous material, tests for bile negative. The indirect Van den Bergh by Doctor Christman of Sacramento showed presence of 1.5 milligrams of bilirubin per 100 cubic centimeters of blood. Under supportive and eliminative treatment, the patient recovered. Most of the symptoms were gone within five days, and the jaundice left in seven days; but the soreness and enlargement of the liver remained three weeks. A urinalysis at the end of ten days showed presence of a few red blood cells, but no other significant findings.

COMMENT

1. Overdoses, and sometimes conservative doses, of santonin may produce serious poisoning, and should therefore be sold only on prescription.

2. Physical condition of the child, as well as age, should be taken into consideration in determining the dosage.

3. The drug should not be given on an empty stomach, as this would favor absorption. A rapidly acting cathartic should not be forgotten, as it would shorten the absorption time.

Fourth Street at D.

FETAL PERITONITIS*

By ALFRED J. SCOTT, JR., M. D.
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IN a recent journal, Abt[1] discusses the causes for fetal peritonitis and reports illustrative cases. He reviews briefly the history of this subject from the time of Simpson in 1838.

ETIOLOGY

The etiologic factors causing fetal peritonitis may be: (1) Inflammatory processes, either bacterial or nonbacterial, possibly transmitted from the mother through the placenta. (2) Syphilis (?). (3) The escape of intestinal contents into the peritoneal cavity. Prior to the fifth month of gestation, the intestinal contents are sterile; but after the fifth month, probably infectious. Aseptic meconium results in a noninfectious peritonitis. The site of the lesion is usually at the lower part of the ileum, cecum or the sigmoid, and the size of the lesion may be from a pin-point to a large rent. (4) Urinary tract malformations may result in peritonitis. (5) Fetal appendicitis may be present. (6) Adhesions and bands may be due to developmental or embryonal rests, or secondary to inflammation. (7) The theory of embryonal hyperplasia of the alimentary tract endoderm. The persistence of these embryonal occlusions results in the congenital type of intestinal atresias and stenoses.

* From the California Babies' Hospital, pediatric department of the California Hospital.

* Read before the Los Angeles Clinical and Pathological Society, March 23, 1933.

SYMPTOMS

There may be death *in utero* with the pathology present or, if the child is born alive, an early death. There is abdominal distention, dilated superficial blood vessels, a certain amount of rigidity, tympany or dullness, dyspnea and varying degrees of cyanosis. Early vomiting of food, later fecal material. There is crying as an evidence of pain, a varying amount of edema of the lower extremities and icterus also. Aterminal rise of temperature occurs. The infant usually nurses well the first few days of life, but the bowels may or may not move. There may be other congenital defects.

REPORT OF CASE

On the service of Dr. Joseph Nicholson, a male was born spontaneously, November 15, 1931, after a 26-hour first-stage and 15-minute second-stage labor. The birth weight was 4 pounds 14 ounces. The infant was about one month premature.

The prenatal history was that of a normal pregnancy with no venereal history, and no known cause for the premature delivery.

The infant's length was 18½ inches, the head 12 inches in circumference, and the shoulders 12½ inches. No gross congenital deformities were noted.

In the placenta there were several infarcts, and evidence of an old hemorrhagic area centrally placed.

From November 15 to 19, inclusive, the baby was fed with breast and bottle, the weight, however, declining to 4 pounds 7½ ounces. On November 19 the weight rose to 4 pounds 10 ounces, which was held until the eighth day.

The stools, from November 16 to 20, were meconium, and the next two days were tinged yellow and green, with green and yellow curds.

On the evening of November 19, pitting edema was noted in the lower extremities and up the thighs, on to the abdomen as far as the umbilicus. The skin was reddened, and the abdominal vessels were prominent. The legs were cold, spastic and extended. Reflexes were present only in the feet. The abdomen was distended and tense, but the cord-stump seemed normal. The upper extremities were spastic, but not edematous. The fingers were fully extended. The heart was normal and the lungs clear. The infant was toxic but not restless, and did not cry easily.

The urinalysis showed faint trace of albumen, a few epithelial cells, leukocytes, and bacteria.

Blood Count.—Hemoglobin, 102 per cent; erythrocytes, 5,392,000; color index, 0.94; leukocytes, 6,100; polymorphonuclears, 64 per cent; and lymphocytes, 33 per cent.

A few myelocytes, and occasional red cell showing basophilic stippling, and occasional nucleated red cell and a slight degree of polychromatophilia. The platelets appeared normal.

The temperature the first four days of life was between 97 and 98 degrees; but on November 19 it was up to 98.6.

When we saw the infant we advised putting it in an incubator, to raise the body temperature, and give only breast milk every four hours, with water between feedings.

On November 20 the infant was considerably warmer and more active, although the legs were still very hard from the edema.

On November 21 there was less edema of the legs, but the scrotum was now markedly edematous. The abdomen was still distended, but not so tense. On November 22 there was slight blood tinge to the stool.

It was suggested to take a roentgenogram, to see if we could learn anything, as we did not consider the child a good surgical risk. The report was: A dilated esophagus, with the stomach of normal size. The heart was normal in size and shape. The diaphragm was of normal level and curvature. There was much free air or gas in the peritoneal cavity, suggesting a perforation of the intestinal tract distal to the duodenum.

The infant died at 3:45 p. m. on November 22, 1931.

Autopsy by Dr. A. H. Zeiler, performed November 22, 1931.—"Body was that of a premature male infant said to be eight days old. The stump of the dried cord was still attached. There were no scars. The abdomen was distended, and a mass could be palpated in the lower half. This mass was globular in shape and seemed to fill most of the lower half of the abdomen and pelvis. The abdomen was found to be the seat of a diffuse peritonitis, with loops of small intestine matted together in the pelvis and covered with yellow fecal material as well as fibrin and pus. The loops of intestine in the upper abdomen were deeply injected and covered with pus, but the fecal matter appeared to be mainly in the pelvic region. The mass that was felt through the abdomen was found to be the matted-together loops of small intestine.

"The umbilicus and round ligament of the liver, as well as the remains of a hypogastric artery, were dissected out, but evidence of infection could not be traced along these structures. The spleen, liver, gallbladder and kidneys, excepting for the peritonitis, were essentially negative.

"On separating the head of the cecum about one centimeter from the base of the appendix, a small pinhead opening was found on the lateral surface. There was an escape of putty-like fecal matter through this area. It was difficult to determine whether this opening occurred postmortem, due to separation of adhesions, or whether it was an antemortem perforation. The dissection was blunt and very gentle, and it seems probable that the opening was antemortem. This was somewhat corroborated by the presence of fecal matter in the pelvis before any dissection and by x-ray films.

"The large intestine was dissected out and found to be the seat of an ulcerative colitis, with the ulcers mainly in the sigmoid and descending colon.

"Thorax: The lungs were well aerated. There was a moderate amount of hypostatic congestion, but no consolidation. The heart and thymus were negative.

"Microscopic: Sections at different levels of the colon revealed an acute ulcerative colitis. Some of the ulcers were membranous in character. They extended down into and through the submucosa. There was a mild acute inflammation in the serous layer. There was no ulceration at the point of perforation, but on either side the mucosa was superficially ulcerated. There was nothing about the histological appearance of the perforation to prove or disprove whether it was ante- or postmortem.

"Anatomical Diagnosis.—(1) Prematurity; (2) acute diffuse peritonitis; (3) acute ulcerative colitis; (4) perforation at the head of the cecum."

SUMMARY AND CONCLUSION

1. A new-born infant is reported with an ulcerated colitis, which ruptured into the peritoneal cavity.

2. The etiologic factor in this case is in doubt, but probably some antenatal condition.

3. Surgery is very unsatisfactory in this condition, and the prognosis is uniformly fatal.

4. No bacteriologic study was made on account of the fecal contamination.†

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REFERENCE

1. Abt, Isaac: Fetal Peritonitis, Clinics of North America, Vol. 15, No. 3, pp. 611 et seq. (Nov.), 1931.

† Our thanks are due Dr. Joseph Nicholson for the privilege of seeing this infant, and for permission to report the findings.